CORRELATION OF ANNULAR MPI RATIO OF TISSUE DOPPLER IMAING (TDI) IN ACUTE INFERIOR WALL MYOCARDIAL INFARCTION WITH CORONARY ANGIOGRAM FOR IDENTIFYING CULPRIT VESSEL

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BONAFIDE CERTIFICATE

This is to certify that the dissertation entitled **"CORRELATION OF ANNULAR MPI RATIO OF TISSUE DOPPLER IMAING (TDI) IN ACUTE INFERIOR WALL MYOCARDIAL INFARCTION WITH CORONARY ANGIOGRAM FOR IDENTIFYING CULPRIT VESSEL"** is bonafide record work done by **Dr.R.R. SARAVANAN** under my direct supervision and guidance, submitted to the Tamil Nadu Dr. M.G.R. Medical University in partial fulfillment of University regulation for **D.M, Branch II –Cardiology**.

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DECLARATION

I, **Dr. R.R. SARAVANAN** solemnly declare that the dissertation titled **"CORRELATION OF ANNULAR MPI RATIO OF TISSUE DOPPLER IMAING (TDI) IN ACUTE INFERIOR WALL MYOCARDIAL INFARCTION WITH CORONARY ANGIOGRAM FOR IDENTIFYING CULPRIT VESSEL"** has been prepared by me. This is submitted to The Tamilnadu Dr. M. G. R. Medical University, Chennai in partial fulfillment of the rules and regulation for the award of D.M. degree Branch – II (Cardiology) to be held in August 2013.

Place : Madurai **Dr. R.R.SARAVANAN**

Date :

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CONTENTS

INTRODUCTION

Inferior wall myocardial infarction in association with right ventricular involvement occurs in 30-50% of patients and is caused mainly by Right Coronary artery involvement.

Tissue Doppler imaging is a technique which measures myocardial velocities quantitatively in both phases of cardiac cycle.

This study was designed to test the usefulness of Ratio of Lateral mitral annular myocardial performance index by lateral Tricuspid annular myocardial performance index in identification of culprit vessel whether right coronary artery or left circumflex.

The culprit artery involved in Acute inferior wall myocardial infarcts is mostly the right coronary artery and next is left circumflex artery and very rarely due to wrap around LAD .

In case of LCx occlusion 30-50% of individuals present with ST elevation in leads II, III, aVF with ST elevation in II >III while rest shows ST depression in V1 to V4. In 38% patients, no significant ST elevation due to LCx occlusion.

In RCA involvement there will be ST elevation in II, III, aVF and ST depression in Lead I and aVL. ST elevation in III more than Lead II denotes RCA involvement with 99% sensitivity and specificity of 100%.

Inferior wall MI spectrum :

IWMI patients may have additional features of right ventricular infarction (RVI) or lateral and posterior wall infarction. They associated with specific hemodynamic perturbations and increased mortality. In addition to LVEF which predicts prognosis in LV infarcts, regions supplied specifically by the RCA to the right ventricle alters that linear relationship also .Atrioventricular (AV) blocks are commonly present with IWMI.Right ventricular infarction present in 10-50% of IWMI and also predicts complications , like inhospital mortality & morbidity.

Nearly half of the patients have associated atrioventricular blocks whereas only 13% has the same without RV involvement.Though LV systolic function is preserved, decreased filling of RV in RVMI contributes to decrement in preload of left ventricle. Both ↓ preload and conduction blocks leads to increased complications. Patients with associated RVMI has

31% mortality, whereas only 6% in patients without RV involvement . RVMI association has increased complications like shock and arrhythmias.

In the days of PCI also, associated RV involvement has 15% increased MACE on comparing to AWMI or IWMI which has only 8%.

Right ventricular infarction associated with inferior wall MI though initially observed about two decades ago, has gained importance only in recent years.

Right ventricular involvement in Inferior wall MI at the initial time of presentation associated with relative risk of inhospital mortality of 7.7 (95% CI, 2.6 to 23) and involves inhospital major complication of 4.7 (2.4 to 9.95%CI).

About 95% of patients without RV involvement at the time of presentation were discharged from hospital, compared to 69 percent of patients complicated with right ventricular MI during initial presentation.

Reflection of right ventricular infarction by imaging in patients denotes significance of therapeutic implication, in patients with inferior wall MI.

Patients with right ventricular involvement were likely to be sensitive to decreased preload and atrioventricular synchrony loss. These two features will lead to initial decrease in right ventricular stroke volume, and later secondarily involves and decreases left ventricular stroke volume also. Elderly age ,female sex, extension into lateral wall, third degree A-V conduction disturbance and rupture of free wall all predicts outcome. Thrombolysis more useful in high risk groups.

Blood supply of Inferior Wall :

RCA supplies 25% of LV inaddition to RV.PDA arises from RCA in 85% of cases. In the remaining 15% LCx is the source of origin. Inferior wall ,basal IVS,PM papillary muscle receives blood supply from RCA.

Inferior and posterior walls of right ventricle is supplied by posterior descending branch of RCA. RCA gives acute marginal branch which supplies right ventricle's lateral wall. But the anterior wall of right ventricle is supplied by two arteries the conus branch of RCA and moderator branch which courses from LAD.

Several studies showed that very proximal right coronary artery involvement result in large infarct.

Infarction size will be more if right ventricular marginal branches are occluded and also absent collaterals from left anterior descending artery.

Clinical manifestations :

1. Suspect RVMI in all inferior wall infarction patients if they have low cardiac output.
2. If Patient presents with symptoms of hypotension.
3. Patient are sensitive to nitrates, diuretics and other preload reducing drugs.
4. Remaining manifestation are Third degree A-V block, Tricuspid valvular regurgitation, shock, rupture of Free wall of right ventricle and lastly cardiac tamponade.

Clinical Signs :

1. The triad is elevated JVP, hypotension, clear lungs fields if there is associated right ventricular infarction.
2. Infrequent clinical signs are RVS3 and RVS4 which are well heard at lower part of left sternal border and augments with inspiration.

3. With monitoring of hemodynamics right sided filling pressures are disproportionately elevated with respect to left side which is the hall mark denotes right ventricular involvement.
4. Earliest abnormalities following occlusion of proximal RCA is impaired relaxation of the right ventricle.

This leads to increased impedence to initial inflow and as seen on to steep rise of pressure volume curve which may lead to increase of RV diastolic pressure and RV dilation.

RA contributes to impaired RV filling also becomes inadequate and leads to depression of RA function and which has fixed stroke volume and ultimately RV output becomes rate dependent.

This may lead to frequently dysrhythmia and chronotrophic incompetence.

Diagnosis :

Electrocardiography :

A standard 12 lead ECG with Additional right sided precordial leads V3R to V6R and also posterior leads V7-V9 should be taken for

identification of infarct related artery. Elevation of ST segment in V4R denotes right ventricular involvement which is also useful to categorize those patients are in high risk group subset.

V4R ST elevation has overall 76% sensitivity and specificity of 78% and gives a diagnostic accuracy of 72%.

Limitations of ECG :

V4R ST elevation has its own limitations which is transient disappears rapidly in less than 10 hours in 50% of patients and it loses its specificity if associated with other heart disease which alters ST segment elevation in V1 like Acute Anterior wall M, Pulmonary embolism, pericarditis and left anterior hemi block.

Ratio of ST elevation is Lead III / Lead II > 1 and S/R wave ratio > 0.33 plus ST depression more than 1 mm in AVL are sensitive& specific markers of RCA occlusion.

LCx lesion should be considered if there is ST elevation of II, III and ST elevation in lead II>III .In some cases ST depression more than 1mm in chest leads V1-V4 alone only present.

Imaging Modalities :

With appropriate clinical setting diagnosis of right ventricular MI with inferior wall can be made with help of non invasive imaging test. Otherwise patient may need invasive hemodynamic monitoring and right hart catheterization.

Echocardiography :

Right ventricular infarction association with inferior wall MI resulting from occlusion of RCA and imaging the right ventricular function is important.

Complex geometry of RV prevents its assessment with conventional echocardiogram .

Right ventricular dilatation abnormalities includes decreased right ventricular wall motion, paradoxical interventricular septal movement and tricuspid regurgitation all gives clue to right ventricle involvement.

The visual assessment alone by echocardiography sometimes leads to under estimation of hypokinesia because of asymmetric thickness of right ventricular wall towards its centre.

Assessment also challenging due to its asynchronous contraction with left ventricle.

Because of these factors limit the validity of usefulness of simple geometric assumptions and limit the use of non invasive techniques like CT and radio nuclide angiography.

Prognostically RVMI association with inferior wall myocardial infarction has 30 day inhospital mortality and morbidity around 30% (13)

So, it has been advised that pulse wave tissue Doppler imaging which is useful to assess left ventricular function also can be used in RV function assessment in inferior wall myocardial infarction patient.

Echo cardiogram shows sensitivity 82% and specificity 93% when right ventricular scintigraphy is cited as reference standard. (14)

Tc^{99} pyrophosphate scintigraphy and gated equilibrium radio nuclide angiography are used in diagnosis of right ventricular infarction noninvasively (15).

In the case of radionuclide study, right ventricle is enlarged and poorly contractile with reduced EF.

With ^{99}Tc study the RV free wall appears as hot which indicates significant infarction.

Hemodynamic Monitoring :

Disproportionately elevated right sided filling pressures in relation to left sided hemodynamics is the hallmark of right ventricular infarction associated with inferior wall myocardial infarction.

Right atrial pressure of more than 10mmHg, right atrial to pulmonary capillary wedge pressure more than 0.8 are accepted as Right ventricular infarction hemodynamic criterias. After adequate volume loading only these values will manifest (16).

Other features includes

- Prominent 'y' descent in JVP
- Inspiratory increase of JVP or right atrial pressure or Kussmal sign positive.

Tissue Doppler Echocardiography

Tissue Doppler imaging is a technique measures velocities of myocardial wall and assess myocardial wall systolic and diastolic movements quantitatively. (17,18)

The ventricular overall function depends on normal contraction of longitudinal and circumferentially oriented myocardial fibers. Quantitation of left ventricular function in longitudinal axis may be clinically relevant, since contraction due to subendocardial fibres is mainly in this direction.

In ischemia which alters specifically subendocardial layers, longitudinal axis will show abnormalities in wall motion very first.

Ischemia of myocardium alters regional contraction and relaxation. Acute ischemia of myocardium leads to loss of contractility, tensile strength and stiffness also.

Tissue Doppler imaging is able to quantify these alterations. During graded ischemia, Derumeaus et al studied the tissue velocity profile pattern in animal model to study that ischemia leads to reduction of Sm and Em rapidly. Good correlation was present between regional myocardial flow and decrease of Sm (19).

Considerable data available on tissue Doppler imaging of RV free wall in diagnosis and prognosis of inferior wall myocardial infarction.

As determined by the position of tricuspid annulus by TDI, myocardial velocities and Myocardial Performance Index(Tei index) of right ventricular function can be assessed.

The measurement of velocities by DTI is a noninvasive also rapid method in assessing right ventricular function in patients with inferior wall myocardial infarction associated with RVMI.

Principles of Tissue Doppler imaging :

With conventional Doppler studies blood flow signals processed and high velocity and low amplitude. Doppler signals reflected by moving blood are measured. whereas high amplitude low velocity signals of myocardial tissues are suppressed.

The new ultrasound method of TDI is based on colour Doppler imaging principles and allows quantification of velocities of intramural myocardium with the use of ultrasound signal consecutive phase shift detection and reflected from the contracting myocardium by changing the thresholding and filtering algorithm to reject low amplitude echoes from blood pool will display regional myocardial velocities. This will improve the ability of measurement of myocardial signals of low velocity which are typically in the range of 0.6 – 24 cm/s.

Conventional Doppler signals are high velocity low amplitude where as myocardial tissue motion are low velocity and high amplitude signals.

Doppler shifts of approximately 40dB higher than blood flow doppler signals are created by tissue motion but velocities exceed rarely 20cm/s.

High pass filters are bypassed with directly entered tissue signal into autocorrelator and gain amplification is reduced to recover low wall motion velocity.

It is important in optimizing the frame rate by narrowing image sector as much as possible to select the appropriate scale of velocity at the time of acquisition of image.

During imaging these parameters are should be optimized since the velocity scale and frame rate modification is not possible in post processing of image analysis.

Modalities of TDI :

Tissue Doppler imaging has three modalities

1. Spectral pulsed Doppler
2. M mode colour Doppler
3. Two dimensional

Spectral pulsed Doppler :

Easiest way to measure myocardial velocity and used for interrogation of myocardial or annular velocities.

It has the advantage of measurement of time intervals and velocities and has an excellent temporal resolution.

Like that of Doppler principle, positive when the tissue velocity moving towards the transducer, but negative when the tissue velocities are moving away from the transducer.

The spectral PW Doppler resolves all peak velocities and provides higher temporal resolution.

The sample volume is placed over the myocardium either epicardium or endocardium.

This modality recorded the low Doppler shift of frequencies which is recorded from the heart wall moving through the sample volume during the cardiac cycle.

It is divided into two parts. Systolic and diastole. Several measurements can be obtained from this.

1. Systolic phase is identified to positive wave (SM) which is preceded by time called as Regional isovolumic contraction.

2. Diastolic phase is complex which comprised of measurements

   a. Regional isovolumic relaxation time

   b. Period of rapid filling characterized by negative were Em

   c. Diastasis

   d. Second negative wave Am due to atrial contraction related filling of ventricles.

Color Doppler :

In color TDI wall motion toward the transducer (Positive velocities) is encoded by red color, where negative velocity due to wall motion away from the transducer encoded by blue color.

The brightest shades on each side of the scale correspond to the highest velocities.

Digital acquisition and storage of color images required for offline post processing analysis.

Endocardial and epicardial layers analysed separately in contrast to the spectral Doppler TDI.

In each phase of cardiac cycle mean and peak velocity, TVI and regional time interval are measured in each segment of myocardium.

High temporal resolution (5-10ms) is present in M mode color encoded TDI.

Slow frame rate limits colour two dimensional imaging. But advances in beam formation technology and parallel processing increased the frame rate to a level required for most cardiac event analysis.

Clinical usefulness of pulsed TDI

Global assessment of LV function

In apical views peak mitral annular velocity used to assess global LV systolic function. Coordination of circumference and longitudinal fibres are necessary for synchronous myocardial contraction and relaxation.

Activation disturbance and vulnerability to myocardial ischemia of longitudinal fibers are related to their subendocardial position.

Since due to relatively fixed epicardial apex shortening of longitudinal fibre draws the A-V ring towards ventricular apex.

This will contribute to fall of LV cavity volume with ejection but increases the left atrial volume at the same time.

During diastole similarly atrioventricular ring backward movement aids the LV filling. Dumesnil et al showed 15-20% decrease in circumferential fiber shortening. In short axis corresponds to 40% decrease of EF by M mode highlighting the longitudinal fibres significant contribution in overall LV function.

Total excursion and peak systolic mitral annular amplitude correlates well with stroke volume of LV with mitral annular descent concept by echocardiography.

Its velocity and amplitude used to calculate EF and corrlelate with radionuclide ventriculography obtained LVEF.

It not only measures and defines the movement of mitral annulus but also gives high temporal relationship.

LV asynergy and infarct regions in previous myocardial infarction patient shows decrease in mitral annular peak systolic descent velocity and corresponding longer time to peak (20-23)

Inotropic stimulation induced alteration in LV contractility are measured by sensitive markers using mitral annular velocity.

Gorcsan et al showed with low dose dobutamine (1mg/kg/mt) significant increase in peak mitral annular velocity and decrease in time to peak velocity correspondingly.

Increase of the velocity in a linear dose dependant manner was observed upto infusion rate of 5 mg / kg /mm. However upto 2 mcg/kg/mm dose does no change in regional peak velocity and upto 3 mc/kg/mm EF and myocardial wall thickening was also not changed.

As a sensitive global systolic function marker, use of mitral annular velocity is supported by these datas even when the definition of endocardial border is suboptimal for measuring Ejection fraction and LV volumes.

This provides importance of mitral annular velocity in terms of velocity amplitude and timing in relation with cardiac cycle.

Ozedemir et al concluded in their study for identifying proximal RCA lesion , the systolic velocity of RV < 12 cm/s and RV MPI > 0.70, are predicts proximal RCA as culprit lesion. (24)

Interestingly MPI(Tei) of lateral mitral annulus divided by MPI(Tei) of lateral tricuspid annulus reliably identifies the culprit lesion.

Normal control group has mean value of 1.08 whereas elevated lateral mitral annular MPI denotes significant ischemic dysfunction in territory of LCx and RV involvement is relatively spared. This index is useful for LCx culprit lesion identification (25).

In the proximal RCA involvement opposite phenomenon occurred because MPI of Lateral Tricuspid Annulus is increased.

Many studies report RV infarction patient has early hemodynamic improvement and RV function recovery even without reperfusion of culprit vessel within 10 days after RV infarction. Recovery of right ventricular ejection fraction was demonstrated by radionuclide ventriculography assessment of RV function.(26)

Assessment of Diastolic function :

Analysis of myocardial motion in diastole with TDI was performed in healthy controls. The PW-TDI shows mirror image of initial inflow with E/A velocity decreasing with age. Differentiation between constrictive pericarditis and restrictive cardiomyopathy, the myocardial velocity measurements was used among the patients with different patterns of myocardial hypertrophy.

Mitral annular E velocity is preload independent diastolic function variable. But subsequent work by many investigators debated the load independent function of Ea.

But it is often necessary in day to day practice to measure with non invasive tests regarding the filling pressures of left ventricle and which is useful in guiding the management of patients with heart diseases.

Ratio of E/Ea was proposed by Nagueh et al as an important tool in left ventricular filling pressure by non invasive assessment methods.

They divided 125 patients into three groups in their study. Left ventricular E/A >1 (normal), E/A < 1 (impaired relaxation) in asymptomatic persons and E/A > 1 (pseudonormal) but with heart failure symptoms. Patients with impaired relaxation and pseudo normal pattern has reduced Ea valve. Pulmonary artery wedge pressure correlates best with E/Ea rate (r-0.87).

In this, second study of nagueh et al pulmonary artery wedge pressure were recorded accurately by using E/Ea ratio, even in the presence of sinus tachycardia.

In their study of 100 cases which included complete merging of E and A inflow velocities in 35 patients. Strongest correlation of E/Ea ratio to pulmonary capillary wedge pressure was found. Cr = 0.86. PCWP = 1.55 + 1.47 E/Ea). Cut off value of E/Ea > 10 predicts PCWP of > 15 mmHg with sensitivity 92% and 80% specificity.

In patients with varying LV diastolic pressures but with normal LV systole function without significant coronary artery disease in 70 hypertensive patients, utility of the deceleration time of mitral annular Ea, Aa time and E/Aa ratio was evaluated by Dagdelen et al in estimation of left ventricular end diastolic pressures.

The sensitivity and specificity values for prediction of left ventricular end diastolic pressure of > 15mmHg were Aa time > 110 ms (71% & 80%), Ea at > 120 ms (88% and 81%) and Ea / Aa (86% and 72%) respectively. (27-31)

Usefulness of TDI in coronary artery disease :

Ischemia of myocardium alters regional contraction and relaxation. Apart from loss of contractility ischemic myocardium also suffers loss of stiffness and tensile strength. Tissue velocity imaging can quantity these alterations.

Tissue velocity profile in an animal model during graded ischemia studied by Derumeaux et al, showed rapid reduction of early diastolic and systolic velocities. 46% reduction of systolic velocity within left anterior descending artery occlusion of 5 seconds. At around 30 seconds systolic waves became negative and peaked at 1 min. This correlates to paradoxical expansion of the same segment. Simultaneous occurrence of diastolic abnormalities was characterized by decrease in Em and increase in Am waves.

Isovolumic relaxation and contraction velocity increased and peaked around 1 minute. Good correlation between regional myocardial flow and decrease of systolic velocity was seen.

Measurement of transmural extent of myocardial necrosis by Pislaru et al in another animal model after LAD artery occlusion for one to two hours and the effect of its on contraction and relaxation of myocardium. Morethan 20% necrosis of thickness of myocardium shows absent isovolumic contraction wave which is preserved in less than 20% necrosis of myocardial thickness. Reperfusion did not improve wall thickening. But positive isovolumic contraction velocities distinguishes segments with different degree of necrosis.

Smaller infarcts associated with hypokinetic or akinetic segments shows preserved isovolumic contraction waves. However it should be remembered that it cannot differentiate ischemia and perfusion induced contractile dysfunction since they resemble each other.

Therefore, through myocardial velocity, estimation of myocardial perfusion is valid only in situations like ongoing ischemia and not following reperfusion.

Tissue Doppler imaging detects minor abnormalities in LV isovolumic relaxation and contraction, also longitudinal shortening in patients like chronic stable angina. For quantifying regional wall motion abnormalities during stress echocardiography TDI has also been used. For detecting wall motion abnormalities the utility of TDI in 60 patients was evaluated by Katz et al. Excluding the apical segments at peak stress for identifying abnormal segments, less than 5.5 cm/s of peak velocity with peak stress correlate with average 96%, 81% and 86% of sensitivity, specificity and accuracy.

TDI also used as accurate tool in dobutamine stress echo cardiography to assess myocardial viability. 23 coronary artery disease patients with resting LV dysfunction was evaluated by Larrazet et al to

assess the concordance between standard echo and using TDI during dobutamine stress echo cardiography and thalium 201 SPECT for assessment of viability. Comparison of standard echo evaluation and TDI during dobutamine stress had sensitivity (60% Vs 82%), equal specificity (100% each) and higher accuracy (74% Vs 94%) for detection of viable myocardium using thallium 201, as the gold standard. Predicting viable myocardium in 56 patients undergoing coronary angioplasty evaluated with conventional dobutamine stress echo cardiography and color tissue Doppler imaging by Nishino et al.

For prediction of recovery of dysfunctional segments Dobutamine TDI alone is used or in combination with conventional stress echocardiography has sensitivity higher and equivalent specificity. For counter balancing the poor agreement of standard echo in evaluation of wall motion, Tissue Doppler Imaging is found to be useful superior technique. In interpreting regional wall motion of basal segments tissue velocity profile is important. Tissue Doppler imaging improves the accuracy of dobutamine stress echocardiography significantly by novices and also by experienced echocardiographer who were not trained formally in stress echocardiography. 77 patients

underwent coronary angiography and stress echocardiography in a study, the baseline accuracy improved by TDI for novice readers from 68% to 76% (p=0.001) for especially basal segments which were scored normal otherwise.

Tissue Doppler imaging in right ventricular infarction :

Right ventricular function was impaired in right ventricular myocardial infarction. Because of complex geometry of right ventricle artery conventional echocardiographic examination is difficult to assess anatomy and function of right ventricle. Asymmetric contraction towards centre of right ventricular wall leads to under estimation of hypokinesia of free wall of right ventricle during visual assessment in most cases. So pulsed wave TDI or color coded TDI which are used frequently for assessing left ventricular function can also be used for assessment of right ventricular function. The longitudinal dynamics of the right ventricle assessed by myocardial velocities obtained from right ventricular free wall using pulsed wave TDI at tricuspid valve annular level in apical four chamber view which is not seen well with visual scoring.

Other than using the TDI method in studies assessing right ventricular function, tricuspid annulus movement represent global function of right ventricle. Systolic tissue velocity obtained by pulsed wave TDI close to tricuspid lateral annulus level of right ventricle free wall in apical four chamber view, using stress echo with dobutamine documents the involvement of significant RCA disease.(32-36)

Systolic velocity of tricuspid annulus decreases in IWMI compared with Anterior Wall Myocardial Infarction and patients with or without RVMI.

REVIEW OF LITERATURE

In acute inferior wall myocardial infarction patients, approximately half of them develops right ventricular myocardial infarction. Major complications and inhospital mortality is high in patients with associated RVMI. Until recent years little clinical attention only received despite serious hemodynamic consequences initially.

Right ventricular infarction markedly contributes to hemodynamic instability, inhospital mortality and atrioventricular conduction block in patients with inferior wall myocardial infarction. The following studies evaluated the usefulness of myocardial tissue velocity in patients with inferior wall myocardial infarctions.

1. A study of IWMI with or without RV involvement – treatment and inhospital course.

Pietr kukla, Darin Dudek, et al

Kardiol pol 2006 : 64 : 583-588

181 consecutive patients with inferior myocardial infarction was evaluated for outcome and clinical course with or without RVMI. Inferior wall MI without RV involvement was diagnosed in 94 (61.9%)

patients and with RV infarction in 65 (35.9%) patients. Patients with RVMI had greater sums of ST elevation in ECG and decreased systolic blood pressure on admission. Mortality was two times lower in patient with RVMI presented within 6 hours of symptoms onset, than those admitted between 6 and 12 hours from the pain onset. Cardiogenic shock was present in 14 patients with RVMI (21.5%) than without RVMI in 4 patients (4.2%). III degree AV block was present in 22 patients with RVMI (33.8%) than 104 patients without RVMI (4.2), they concluded inhospital mortality in patients with RVMI is higher than those with isolated IWMI. Those patients with cardiogenic shock and RVI have high inhospital mortality.

2. A study of Diagnosis and early complications of right ventricular infarction. Garty I, et al . Eur J Nucl 1984 : 9 (10) : 453.

The prospective study evaluated sixty patients with acute myocardial infarction. Of these inferior AMI were 40 patients and 20 patients were anterior AMI. 50% of IWMI 20 patients had associated RVMI with evidence of V4R ST elevation of 0.1 mV. The RVEF was significantly decreased in patients with RVMI on comparing with other

groups (Mean 27%, versus 57%). 16 patients of RVMI (80%) had various complications during the hospital stay versus only 9 patients (45%) with only inferior AMI without right ventricular involvement. Conduction disturbance were most common (7 of 20 versus 2 of 20).

It stresses the importance of prognosis in early diagnosis of RVMI.

3. A study of Diagnosis and prognosis of right ventricular infarction by Rodriguez EA, Dewhurst NG,et al.

Br. Heart J 1986 Jul : 56(1) : 19-26.

The values of non invasive methods like serial ECG, radio nuclide ventriculography, pyrophosphate scintigraphy and cross sectional echocardiography compared in 51 consecutive inferior wall myocardial infarction patients. RVEF by echocardiography methods correlated poorly with radionuclide methods.

Poor RV function was demonstrated by increased uptake of radioactivity on pyrophosphate scintigraphy by RV but negative scan did not exclude dysfunction of right ventricle territory.

Considerable morbidity and mortality was associated with profound right ventricular dysfunction in acute inferior wall infarction patients. V4R ST elevation predicts right ventricular involvement but it is not specific and leads to over diagnosis. So ST elevation in V4R lead, in diagnosis of RVMI has its own limitations. The ST elevation in V4R is transient and disappears in half of patients within 10 hours. ST elevation in V4R identifies high risk subset in patients with inferior wall myocardial infarction (Robalini et al 1990)8

4. A study of The diagnostic value of 12 lead ECG in predicting the infarct related artery and right ventricular involvement in acute IWMI . Kabake, G. Yiblira A, et al . hacetheps university, Dept of cardiology, Ankara, Turkey. Ann Non invasive electrocardiol 2001, July : 6 (3) : 229-35.

The study evaluated the value of presentation ECG, 24 hour ECG in defining infarct related artery and RV involvement in patients with acute inferior wall myocardial infarction. 149 patients with acute inferior MI were included. Validity of ECG criteria for diagnosis of IRA in RVMI is investigated.

Q wave width in III > II supports RCA as IRA. The sensitivity is 60% where as specificity is 61%. On admission ECG, V1 ST elevation without ST elevation in V2 has sensitivity 63% and specificity 99% inpatients to diagnosis RVMI. 12 lead ECG diagnosis of RVMI is questionable despite moderate sensitivity and high specificity.

5. A study of Identification of culprit vessel involved in acute IWMI using electrocardiographic criteria

E Bayram and C Atalay et al. The journal of International Medical research 2004 ; 32 : 39-44.

73 patients with acute inferior wall myocardial infarction was tested for ECG changes to identify the right coronary artery or LCx as culprit vessel. ST elevates in lead III more than lead II has sensitivity 86% and specificity of 94% in implicating RCA s culprit vessel, and an S/R wave ratio > 0.33 with ST segment depression more than 1 mm in aVL sensitivity increased to 92% specificity remains same 94%.

6. A study of TDI in right ventricular myocardial infarction

Pulse wave TDI and color coded TDI which was used for assessment of LV function may also be used for RV function assessment. In the apical 4 chamber view myocardial tissue velocity at the level of lateral annulus of tricuspid valve of RV free wall shows , the right ventricle longitudinal dynamics which were not well seen in visual scoring analysis.

Studies other than pulse wave TDI which assesses the right ventricular function also represent the global right ventricular function by the tricuspid anuulus movement. Assessment of systolic velocity of tricuspid lateral annulus of RV free wall identifies significant involvement of right coronary artery diseases in A4C view during stress echo cardiography using dobutamine. (32-36).

The following studies were conducted for assessment of inferior wall with or without right ventricular infarction by using Tissue Doppler imaging.

7). A study of assessment of RV function by PW-TDI in patients with acute MI. Warseem Amir, Sameh Bakhoum, et al. Heart Mirror J 2008 ; 2(1) : 6-17.

40 patients divided into four groups. I. Anterior wall MI, II) Inferior wall MI without RV involvement, III) Inferior wall MI with evidence of RV infarction, IV) Control groups and both conventional echo cardiogram and pulsed were TDI was performed.

TDI indices like, SM, Em, Am, peak early diastolic velocity E wave , A wave of late diastolic velocity, ICRT, IVCT are all measured. Basal RV free wall, mid RV free wall lateral annulur of tricuspid valve in group I showed similar to controls whereas TDI of medial annulus of mitral valve, Basal IVS, Mid IVS showed statistically significant difference from controls.

Group II and III showed similar to control regarding lateral mitral annular TDI value, but statistically significant difference was noted in group III those with RV involvement in TDI value of Basal RV free wall, mid RV free wall, lateral annulus of tricuspid valve.

They concluded Sm of lateral tricuspid annulus, mid and basal RV free wall as sensitive tool to diagnose RV infarction. The cut off

value of 11.1 cm/s of lateral tricuspid annulus peak systolic velocity used for separation of patients with on without RV infarction and has sensitivity of 98% specificity of 90%.

8. A study of Tricuspid Annular motion in RCA related acute IWMI with or without RV involvement. Sendar Bayata et al , Anadolu kardio Drug 2011 ; 11 :504-8.

Thirty one patients of inferior wall myocardial infarction with or without RV infarction were included in the study.

Lateral Tricuspid annular movement by PW-TDI and TAPSE also measured.

In patients with RVMI, systolic velocity Sm and TAPSE increased significantly after thrombolysis compared with prethrombolysis.

TAPSE and significantly high annular velocities were present in patients without associated RVMI than patients with RVMI (21.6+ 2.1 vs 16.2 + 2.0, 136.1 + 8.8 vs 110 + 12.6 for Sm)

They concluded echocardiographic detection of right ventricular involvement in patients with IWMI involving right coronary artery by

measuring tricuspid annular movement and velocity measurement by pulse wave TDI.

9. A study of Assessment of RV function by TDI in patients with Acute MI. Pramod Jaiswal, Jaishankar K et al International centre for cardiothoracic and vascular sciences, Chennai.

10 patients with first acute MI (Inferior 6, Anterior 4) and 4 control were included. Since right ventricular function was not studied widely after an acute infarction the study was designed by measuring tricuspid annular motion and tricuspid annular velocity to assess RV function.

The tricuspid annular motion of inferior wall MI patients on compared to controls was reduced (19.2 vs 24 mm). The tricuspid annulus peak systolic velocity was reduced in significantly in IWMI compared to controls (12 vs 14.2 cm/s) also the tricuspid annular motion was significantly lower in patients with RVM1 than without RVMI (16.8 and 21.6 mm, $P < 0.01$). RV infarction associated patients had significantly decreased peak tricuspid annular systolic velocity, also decreased early diastolic velocities.

So RV function in association with IWMI patients can be assessed by measuring the Tricuspid annular motion and velocity.

10. A study of TDI of the tricuspid annulus in the detection of RVMI in acute IWMI. H. Dokainish, K Gin K, et al, The vancouver, British Columbia Canada Cv congress 2003.

In acute IWMI the tricuspid annulus TDI was used to detect right ventricular infarction in this study.

Totally 50 patients were enrolled in the study.Occlusion proximal to first RV branch in 44% (22 patients) where as 28 patients had occlusion distal to first RV branch or in other than RCA.

Univariate predictors of RVMI includes raised JVP > 4 cm,

V4R ST elevation, RV dimension, abnormal RV function by visual analysis and tricuspid annular systolic velocity. Only tricuspid annular velocity and RV dimension predicted RVMI on logistic regression analysis in comparison all significant univariate variables.

On considering various clinical and echocardiographic variables, reduced systolic velocity of Tricuspid annulus by TDI and RV

dimension are multivariate RVMI predictors in patients with first ST elevation IWMI presentation.

11. A study of Color TDI evaluation of right ventricular function in patients with RVMI. Oguzhan A, AbaciA, et al , Department of cardiology, Erciyes University school of medicine, Kayser, Turkey cardiology 2003 ; 100 (1) 41-6.

The study evaluated usefulness of color TDI in 35 patients with IWMI to determine RV function. Twenty healthy subjects served as controls. In inferior wall MI with RV infarction had significantly reduced systolic and early diastolic velocity lateral tricuspid annulus compared to healthy subjects (7.8 ± 1 vs 11 ± 2 cm/s $p < 0.002$) and patients with inferior wall MI without evidence of RV infarction 7.8 ± 1 Vs 10 ± 1 cm/s, $P < 0.002$)

Am value is not differed between the groups. RV free wall systolic and early diastolic velocity is also decreased significantly in 10 patients with RV infarction than those with healthy controls (7±1 vs 8.7 ± 1 cm/s $p < 0.01$, 6.3 ± 2 vs 8.7 ± 2 cm/s $p < 0.05$ respectively)

and inferior wall MI without RV infarction. (7±1 vs 9±2 cm/s $p< 0.01$, 6.3 ±2 vs 8.3 ± 2 cm/s $p < 0.05$ respectively.

So colour DTI by measuring tricuspid annular and RV free wall velocities used to assess RV function in patients with RV infarction.

12. A study on RV diastolic function Assessment by TDI in patients with Acute RVMI.

Mohammed Mukhani, et al, Cardiology dept, Royal Hospital, Muscat, Oman. Echocardiography, 2010, 27 : 539-543.

30 patients with IWMI compared with 20 age matched controls and divided into with or without RVMI based upon standard ECG criteria. Peak systolic Sm, Peak early Em, late diastolic Am, Em/Am ratio and time to Sm along with time to Em were measured in A4C view at the level of lateral tricuspid annulus by using TDI. Sm, Em and Em/Am significantly reduced in patients with RVMI compared without RVMI and healthy controls. Q – Em interval was significantly prolonged.

SM 11.1 ± 2.9 vs 14 ± 1.9 and 14.6 ± 2.1 $p < 0.01$

Em 9.2 ± 3.5 vs 12.9 ± 3 and 14.0 ± 2 cm/s $p < 0.01$

Em/Am ratio 0.53 ± 0.2 vs 0.78 ± 0.19 and 0.8 ± 0.3 $p<0.01$

RVMI was associated with abnormal RV TDI diastolic parameters. This is a simple method used for assessment of RV diastolic function in RVMI patients.

13. A study of New parameters in Identification RVMI and proximal RCA lesion. Kurtulus Ozdemir, Bulent B et al Chest, 2003; 124 : 219-226.

The study prospectively assessed 60 patients who had first acute inferior MI / Mean age 54± 6 years. RVMI associated with > 0.1 mv ST elevation in V4 R lead. The diastolic parameters of Sm, Em, Am, IVCT, IVRT of the lateral tricuspid annulus was recorded by using pulsed TDI in apical 4 chamber view.

They were grouped according to infarct related artery by angiography into three groups.

Group I Proximal RCA

Group II Distal RCA

Group III LCx

Proximal RCA was infarct related artery in 27 patients and RVMI was detected in Sixteen patients. The RV Sm was significantly low in patient's with RVMI than those without RVMI in group I and in groups II and III. Sensitivity and specificity of Sm < 12 cm/s were 81% and 82% respectively in diagnosing RVMI and diagnosis of proximal RCA as culprit vessel with these value, having sensitivity 63% and specificity 88% respectively. MPI is high in same patients with RVMI than without RVMI respectively $P<0.001$.

Sensitivity of 94% and specificity of 80% were calculated when MPI of > 0.70 was used in diagnosing RVMI, and sensitivity of 78%, specificity of 91% respectively in the diagnosis of infarct related artery as proximal RCA.

14. A study about RVMI and TDI – Insights from acute IWMI. After PCI. Shih – Huang Hsiao MD, Kwanhan chion MD, et al. Doi Circulation Journal 201, 10, 1253 / circjh CJ – 10 -0302.

165 patients were evaluated and received primary PCI for acute IWMI were enrolled.

By using TDI, IVCT, IVRT and ejection time, Sm, Em & Am were obtained to calculate MPI in placing Doppler sample volume at lateral tricuspid annulus level of RV free wall and lateral mitral annulus level of LV free wall.

RV - Sm is lower in proximal RCA culprit lesion and also MPI of lateral tricuspid annulus becomes more increased because of IVCT and IVRT were significantly increased in the same group.

But if the involved vessel is LCx it has highly increased MPI of lateral mitral annulus. Lateral mitral annular MPI / lateral tricuspid annulus MPI is used in identifying culprit lesions reasonably.

If proximal RCA was culprit this index is low but when culprit lesion is in the LCx this index is increased due to incremental increase in lateral mitral annular MPI. Lateral mitral annulus MPI / Lateral tricuspid. Annular MPI > 1.06 predicts LCX as culprit vessel.

But for RVI identification the lateral tricuspid annular MPI best indicator, RVMPI > 0.42

With sensitivity of 82% and specificity of 76% IVCT, IVRT of right ventricle and MPI of lateral mitral annulus / MPI of lateral tricuspid annulus providing variant power for detection of RVMI.

15. A study of lateral tricuspid annulus Tei index in RVMI. Chockalingam A, Gnanavelu G, Alagesan R, et al Echocardiography : 2004, August, 21 (6), 487-494.

36 patients with RVMI diagnosed by > 1 mm ST elevation in V3R – V5R were included and 63 patients without RVMI was constituted as control group. Normal range of MPI was derived from 50 age matched healthy controls. RVMPI was increased to Mean 0.53 ± 0.22 in RVMI (Normal MPI 0.20 ± 0.05 $p < 0.01$ inferior wall MI without RVMI did not show elevation of MPI significantly (0.21±0.17 p value N).

Repeat MPI after 5 days shows dramatic reduction (0.23 ±0.1L, $p < 0.001$) irrespective of thrombolysis. 82% sensitivity and 95% specificity was present when RVMPI > 0.3 for the diagnosis of RVMI in presence of inferior wall MI. So, MPI can diagnose RV infarction reliably and can be used to quantity dysfunction of RV and improvements in RV function.

AIM

To assess the usefulness of myocardial performance index ratio with pulsed TDI in patients with IWMI to identify the localization of culprit vessel.

METHODOLOGY

Study subjects :

50 patients with acute IWMI presented first time and admitted in intensive coronary care unit of Dept. of cardiology, Govt. Rajaji Hospital, Madurai were studied. Out of these 37 were men, 13 were women. 43 patients received thrombolysis as initial part of treatment. 7 were not thrombolysed due to delay in median time and presented after 12 hours of chest pain onset. Patients were matched in respect to age, thrombolysis status, diabetes. This is a prospective study with age between 50±8 years. There were admitted with acute onset of chest pain within previous 24 hours. Standard 12 lead ECG, V3R-V6R and leads V7-V9 were recorded. MI was diagnosed as suggested by symptoms of ischemia, ECG changes of ST elevation more than 1 mm in II, III aVF and CK-MB value of more than two times reference value.

Localisation was determined by ECG evidence of ST elevation III > II in RCA and ST in V4R 0.5mm in associated RVMI. ST elevation in II > III , ST elevation in V7-V9 and also ST depression of V1-V4 > 1 mm in addition determining the LCx lesion.

Accordingly RVMI was identified in 24 patients and LCx was identified in 8 patients based on surface ECG. The study was a prospective study planned from December 2012 to May 2013.

Echo cardiography :

Philips IEE 33 cardiac ultrasonagraphy with variable frequency phased array transducer with TDI capabilities was used for the study. Two dimensional color Doppler and pulse wave Doppler were performed 24 hours from the time of admission.

All the echocardiographic measurement were taken as per the recommendation of American Echocardiography Association. LVEF was calculated using Simpsons method.

Doppler echocardiography :

Doppler recordings were measured by placing the pulsed sample volume at the mitral leaflet tips in apical four chamber views. The filing velocity (E), filling velocity (A) and dT of E wave were all measured E/A ratio was calculated.

If tricuspid regurgitation was present, pulmonary artery systolic pressure was calculated by adding pressure difference between RV& RA to right atrial pressure determined by continuous wave Doppler.

TDI based measurement of Annular velocity

After activation of TDI of cardiac ultrasonographic unit PW- TDI images were acquired. Low wall filter settings (ie. 50Hz) used to reduce the noise and optimized gains to terminate the signals formed by transmitral flow were used to record the images using TDI. A 2 to 5 mm sample volume was used. TDI cursor was placed in RV free wall at lateral tricuspid annulus level and in LV free wall at the level of lateral mitral annulus level and aligned in such a way that the annulus moving along the sample volume line in apical four chamber view.

A major systolic myocardial tissue velocity was recorded with annulus movement toward apex of heart during systole. During diastole due to the movement of annulus toward base two major negative velocities were recorded. They were as follows.

Em During the early diastolic phase myocardial velocity

Am Late diastolic phase myocardial velocity

Ejection time was measured from the duration of Sm. IVRT was measured from time measured between ending of Sm and the beginning of Em. IVCT was measured from end of Am and beginning of Sm.

Myocardial performance of Tricuspid annulus was calculated as

IVRT + IVCT / ET by the values obtained from RV free wall at the level of the lateral tricuspid annulus.

Myocardial performance index of Mitral annulus was calculated in a similar way like previously but by placing the sample volume at LV free wall at the level of lateral mitral annulus. Doppler velocity range -20 to 20 cm/s was selected in this study and velocities measured online at a sweep speed of 50mm/s. Average of three consecutive cycles were used to calculate Doppler echocardiographic parameters...

Coronary angiography :

Coronary angiography was performed in mean window period of 12-36 hours. Total or > 70% occlusion of coronary artery supplying the asymmetric field was accepted as infarct related artery defining feature. Patients were divided into two groups group I has occlusion in right coronary artery and subdivided into proximal to RV branch or distal to major RV branch. Group II has occlusion in left circumflex or one of its dominant OM branch which is large.

Patient in whom infarct related artery was not able to identify was excluded.

Exclusion criterias :

1) Concomittent involvement of kidney Disease and elevated serum creatinine >1.7 mg%
2) Those who had documented h/o previous MI
2) Those who are Not willing for Coronary angiogram.
4) Associated Comorbid conditions like Liver diseases.

Using Judkins technique coronary angiogram was performed and images taken with standard cineangiographic system in multiple views. Luminal narrowing at diameter was graded 0, < 50, 70, 90 and 100%. More than 70% localized luminal narrowing was taken as significant anatomical stenosis.

Statistical analysis :

The data were given as the mean ± SD. An analysis of variance test utilized for the comparison of results between the groups and Pearson Correlation was performed. For unpaired groups comparison One way ANOVA was used. $P < 0.05$ p value was accepted as statistically significant.

The sensitivity, specificity value by using standard formulas were used for calculation. The variability was defined as the difference of percentage between two different measurement divided by the mean of the measurements. (14)

RESULTS

Table – 1

Age distribution

Age in years	PRCA	DRCA / LCX
≤ 50 (8)	4	4
> 50 (42)	20	22
Total	24	26

Table – 2

IRA vs MPI RATIO

IRA	Mean of MPI RATIO	S.D	P value
PRCA (24)	0.772	0.377	< 0.001
DRCA/LCX (26)	1.131	0.323	Significant

MPI Ratio	< 1.06	> 1.06
PRCA	22	2
LCx	3	5

Sensitivity - 75%

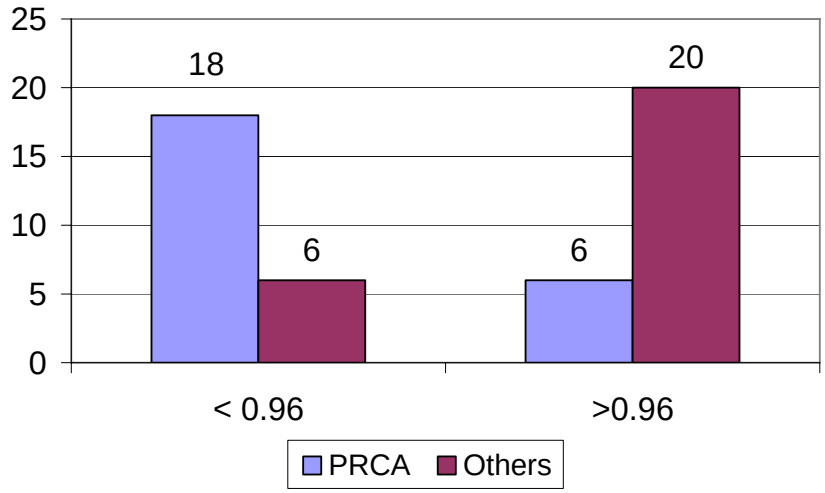
Specificity - 81%

MPI Ratio	< 0.96	>0.96
PRCA	18	6
Others	6	20

Sensitivity - 75%

Specificity - 77%

IRA VS MPI RATIO



IRA VS MPI RATIO

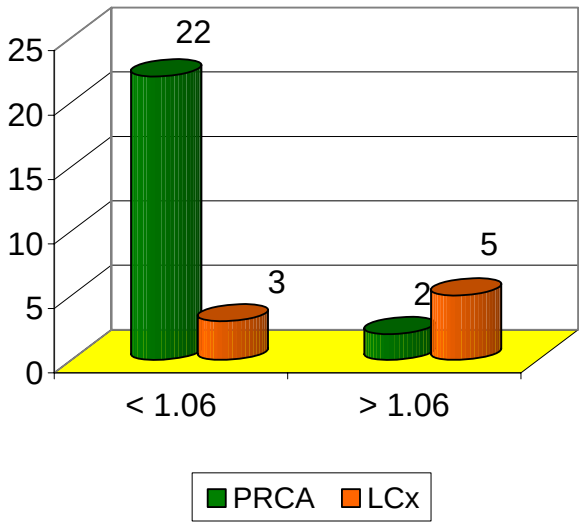


Table – 3

IRA vs RV MPI

IRA	Mean of RV MPI	S.D	P value
PRCA (24)	0.66	0.22	< 0.001
DRCA/LCX (26)	0.44	0.11	Significant

IRA vs RV MPI

MPI Ratio	> 0.42	< 0.42
PRCA	18	6
Others	7	19

Sensitivity - 72%

Specificity - 76%

RV MPI denotes IRA

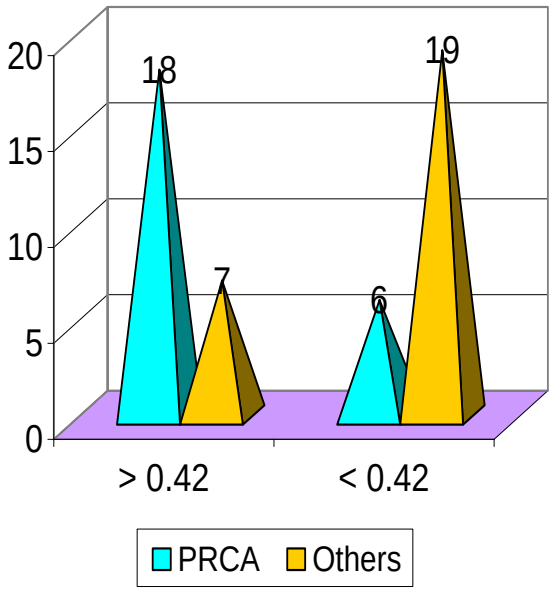


Table – 4

IRA vs RvSm

IRA	Mean of RVSm	S.D	Correlation value
PRCA (24)	9.21	1.48	0.456
DRCA/LCX (26)	9.66	2.05	

IRA vs RVSm

RVSm	< 11 cm/s	> 11cm/s
PRCA	21	3
LCx	8	18

Sensitivity - 72%

Specificity - 85%

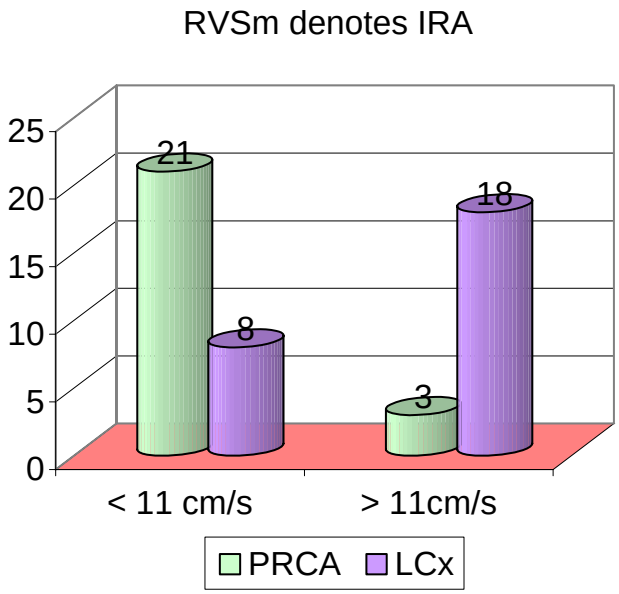

RVSm denotes IRA
25
20
15
10
5
0
21
8
3
18
< 11 cm/s
> 11cm/s
PRCA
LCx

Table – 5

IRA vs IVRT

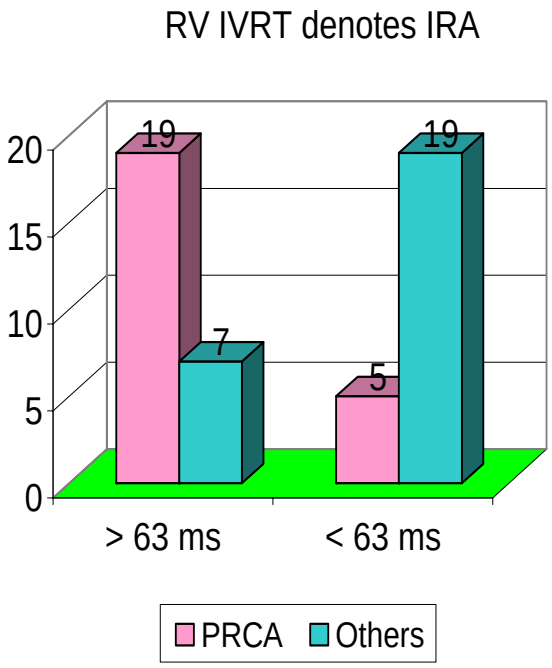
IRA	Mean of IVRT	S.D	P value
PRCA (24)	91.46	41.16	0.364
DRCA/LCX (26)	82.11	30.61	

IRA vs RV IVRT

MPI Ratio	> 63 ms	< 63 ms
PRCA	19	5
Others	7	19

Sensitivity - 73%

Specificity - 79%


RV IVRT denotes IRA
20
15
10
5
0
19
7
5
19
> 63 ms
< 63 ms
PRCA
Others

The clinical parameter in the both groups of infarct related artery were comparable. Patients age matched between the groups.(Table.1)

Whether single or multiple vessel disease detected angiographically and % of culprit vessel occlusion,thrombolysis status,killip class were comparable between the patients. Tricuspid regurgitation was higher in patients with RVMI than without RVMI also SBP was lower in RVMI group.Normal MPI values taken from previous studies.

Annulus velocities and MPI by TDI measurement :

The MPI ratio was less than < 0.96 in patients with associated RVMI and proximal RCA occlusions. Out of 24 patients with proximal RCA occlusion 18 patients had MPI Ratio of <0.96. (P value <0.001). The values of Sm, Em taken from lateral tricuspid annulus was reduced in patients with associated RVMI due to proximal RCA occlusion. (Table.2)

Whereas the Sm, Em, values of lateral mitral annulus were decreased in LCx lesion and IVCT and ICRT are slightly prolonged if the infarct related artery is LCx. The MPI calculated from lateral mitral

annulus was significantly higher in inferior wall myocardial infarction if the IRA is left circumflex system.

The lateral mitral annulus MPI/Lateral tricuspid annulus MPI is more than > 1.06 in LCx culprit vessel. (Table – 2)

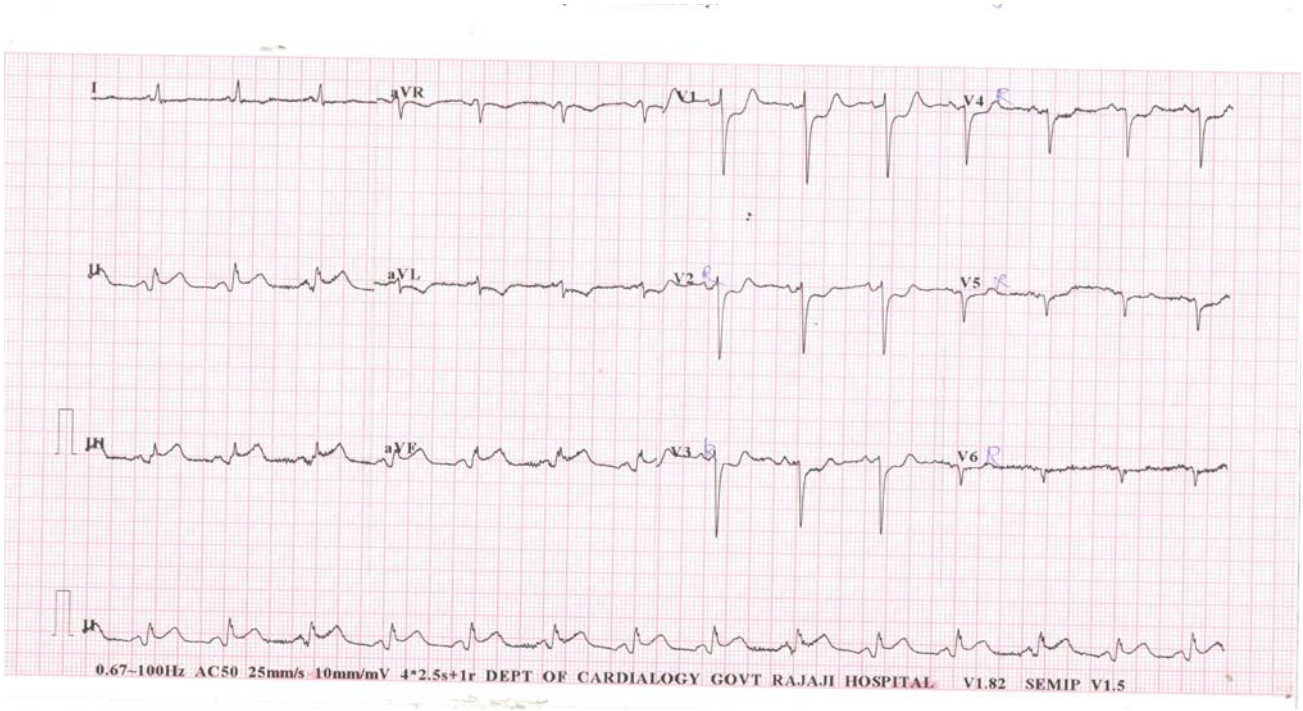
For identification of RV infarction and proximal RCA occlusion MPI of lateral tricuspid annulus of > 0.40 is more useful

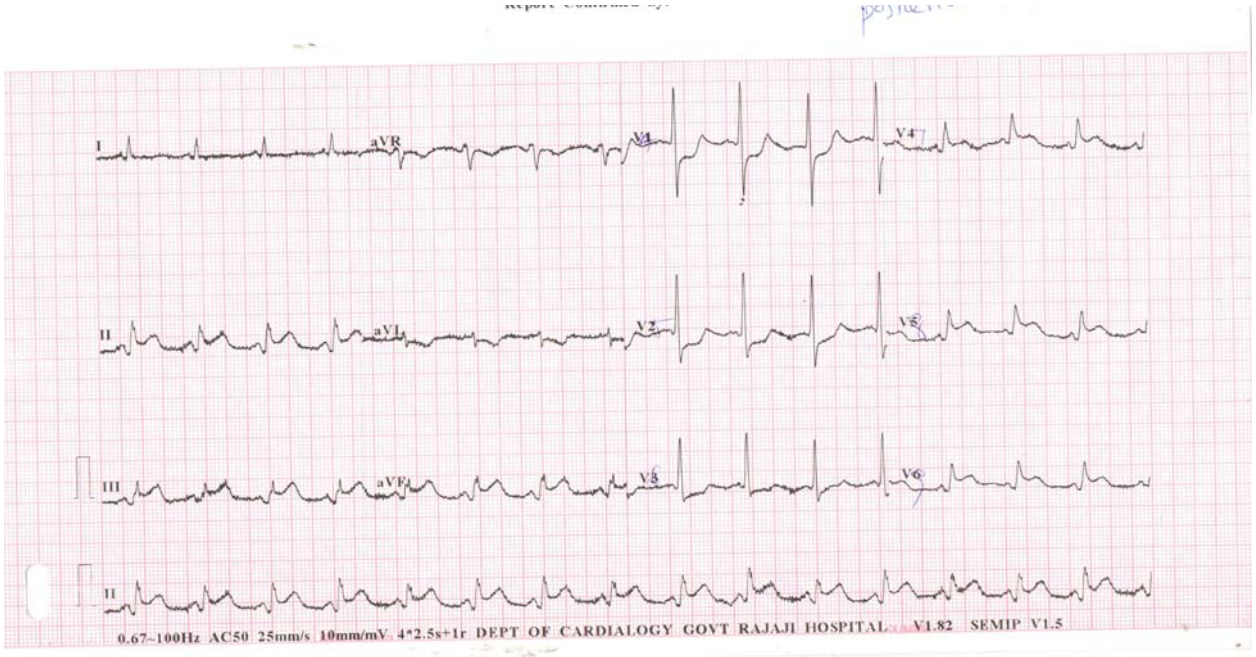
In comparison with patients of IWMI associated with RVMI based on infarct related artery the RVMPI value was increased>0.40 those with RVMI and occlusion of RCA proximally than distal RCA. The RV MPI calculated from lateral tricuspid annulus in patients with RVMI is increased. (Table.3)

Reduced systolic velocity of tricuspid annulus(Sm) provides variant power in assessment of RVI.In patients with proximal RCA lesion Sm values were significantly reduced to <11cm/s.(Table.4)

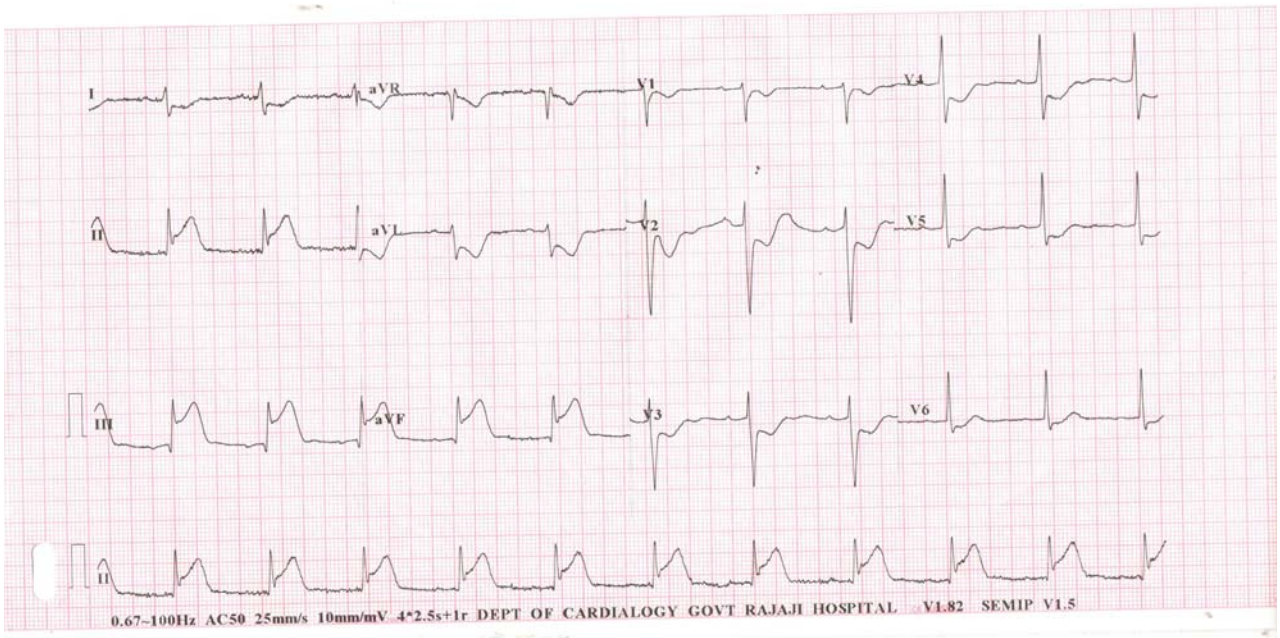
IVRT values were significantly prolonged in patients with RVMI. RV IVRT value >63ms present in 6 out of 24 Proximal RCA occlusion patients.(Table.5)

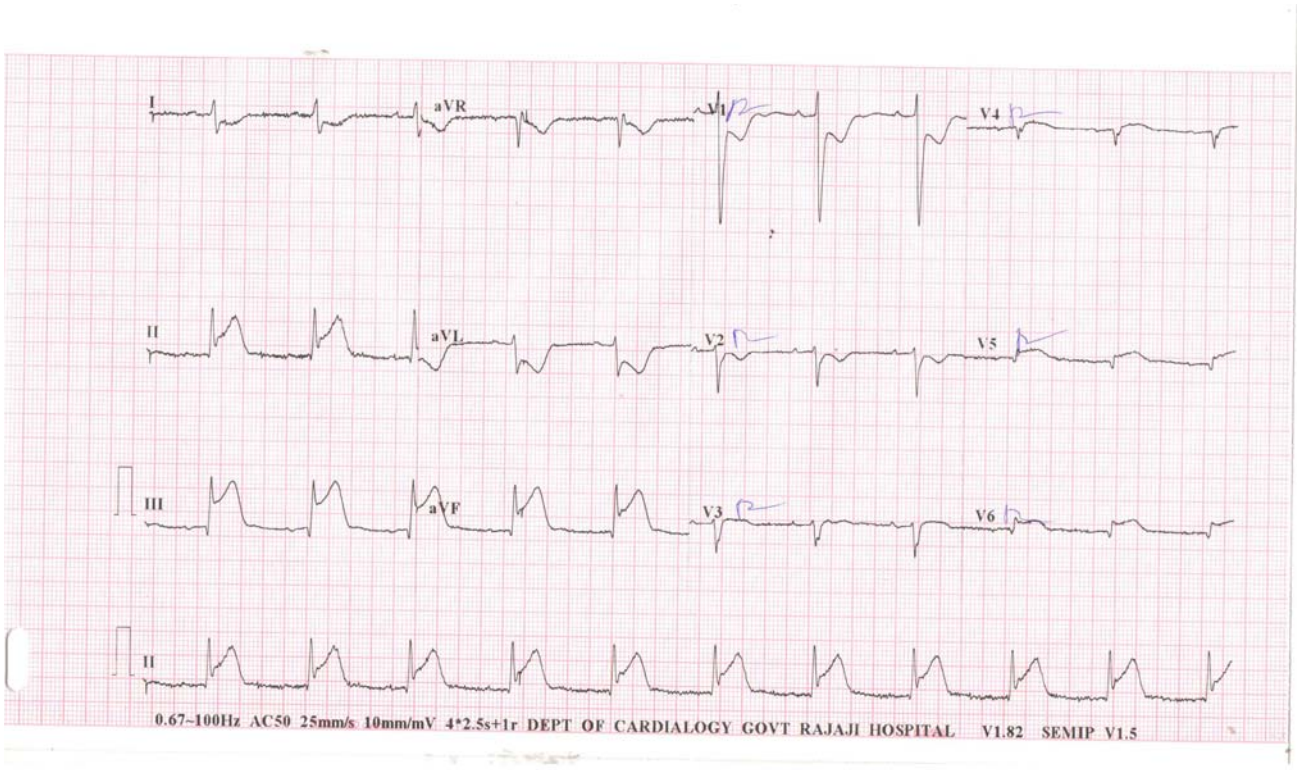
ECG SHOWS IWMI DUE TO LCX OCCLUSION



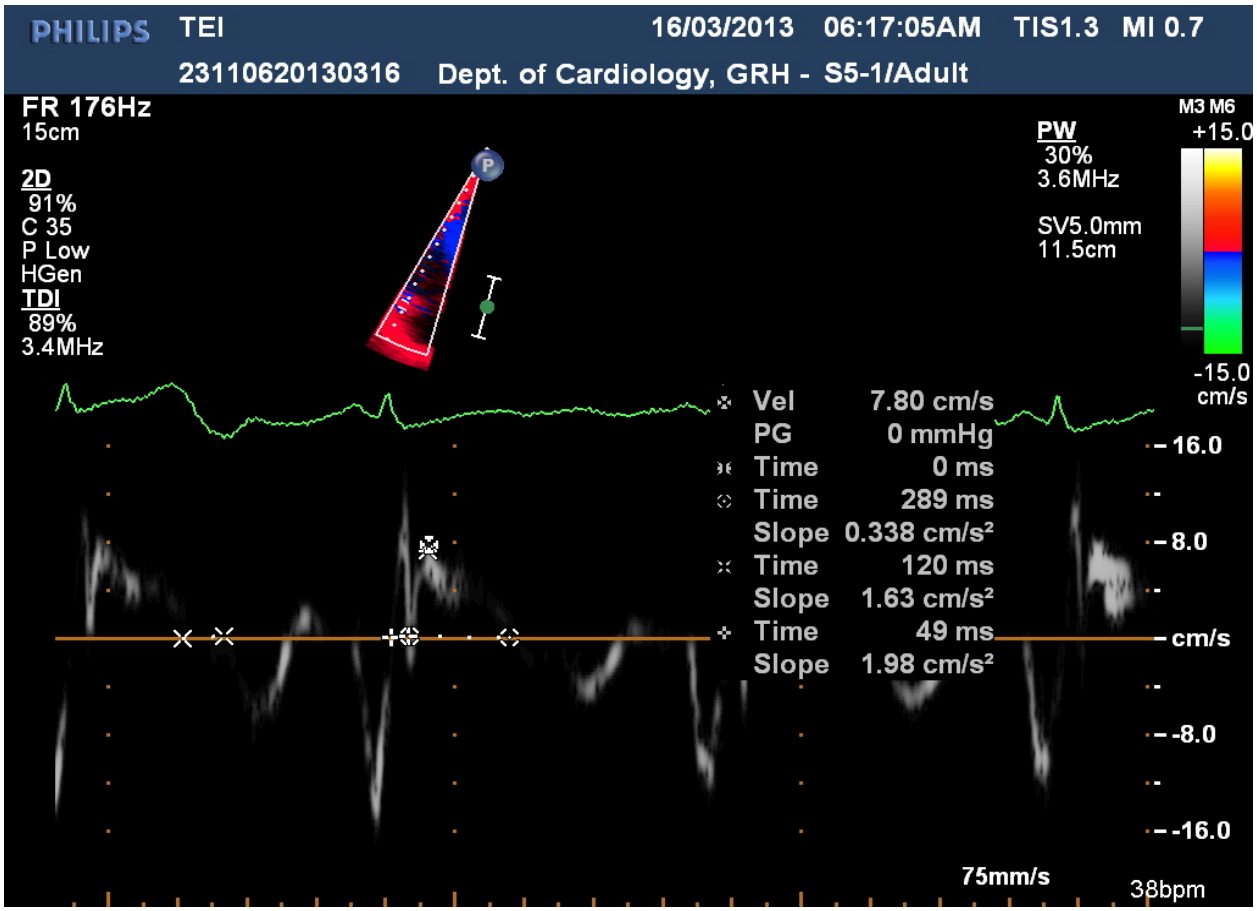


ECG SHOWS IWMI DUE TO RCA OCCLUSION

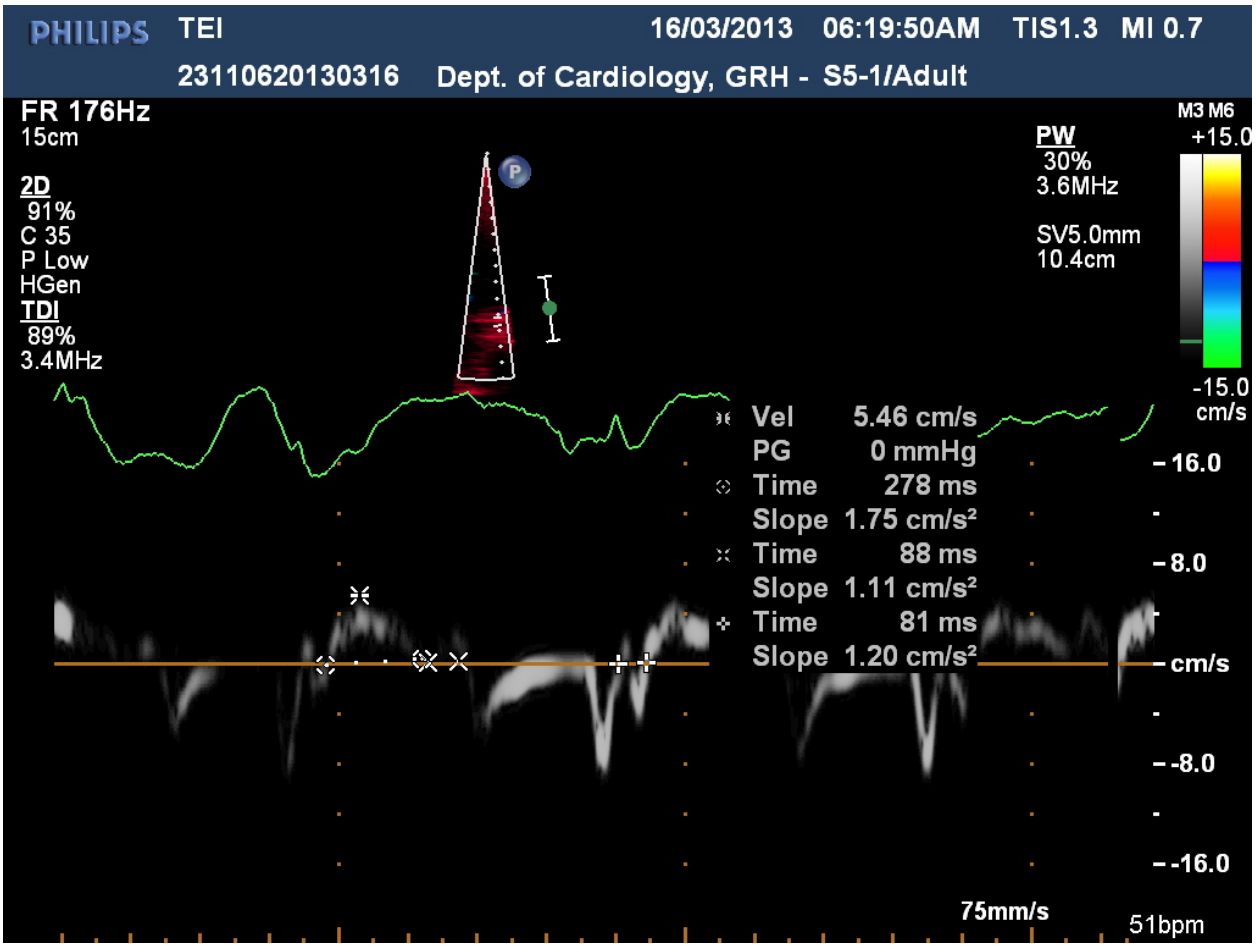




TISSUE DOPPLER IMAGING OF LATERAL TRICUSPID ANNULUS

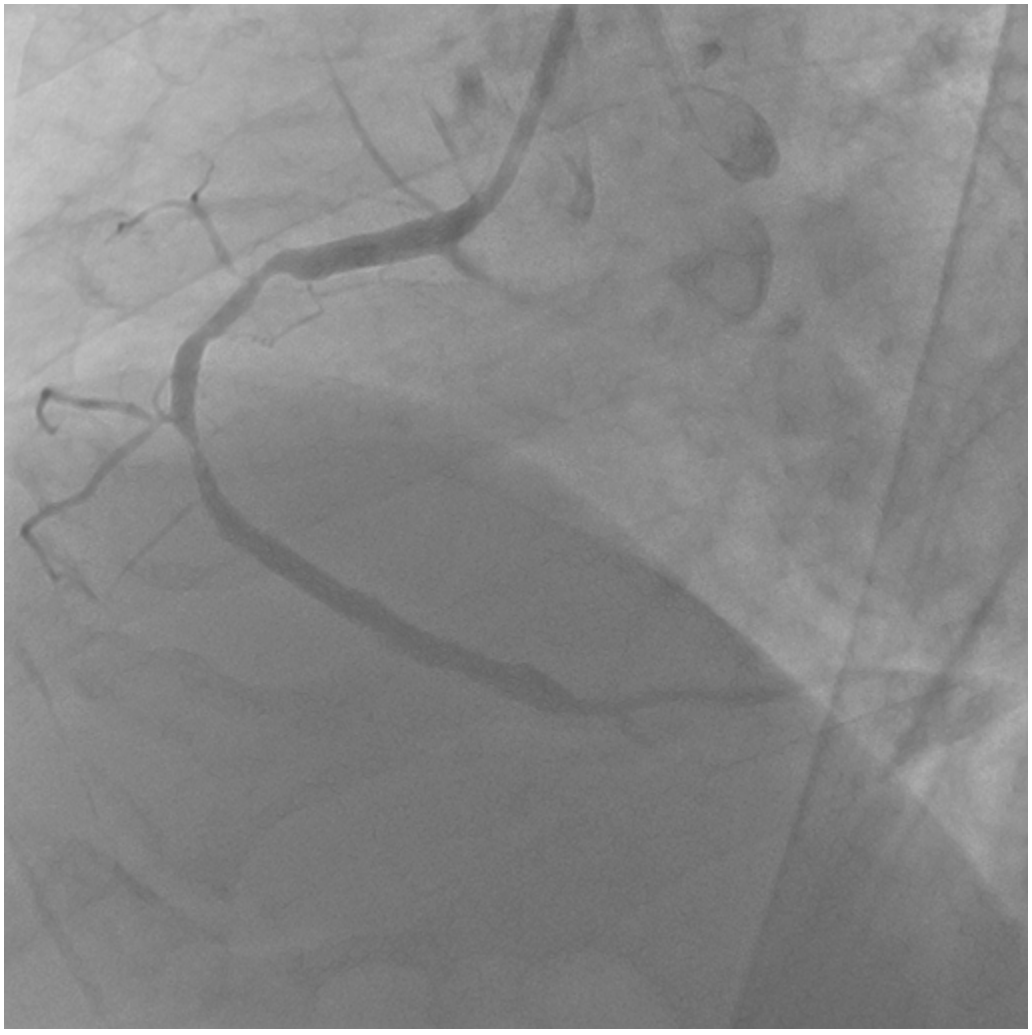


TISSUE DOPPLER IMAGING OF LATERAL MITRAL ANNULUS



ANGIOGRAPHIC IMAGE OF LCX OCCLUSION

ANGIOGRAPHIC IMAGE OF RCA OCCLUSION



PHILIPS iE 33 ECHO CARDIOGRAM

DISCUSSION

Inferior wall MI due to RCA occlusion affects RV function and is also considered as a prognostic indicator. (37,38)

Inferior wall myocardial infarction in 10-50% of cases was associated with right ventricular myocardial infarction. Hemodynamic instability was more if it was associated with RVMI and arrhythmias, inhospital mortality was more if IWMI also associated with RVMI.

Because of complex geometry and anatomy, conventional echo was not very much useful. In case of inferior wall myocardial infarction if the occlusion was due to left circumflex system left ventricular free wall was also involved. RVMI associated with IWMI usually did not alter LV free wall movement markedly.

Visual assessment of hypokinesia of RV free wall leads to under estimation due to asymmetric contraction of right ventricle toward its centre.

So it has been suggested that pulse wave TDI or color coded TDI would be useful in assessing both left ventricular and right ventricular function reliably.

TDI measurement at lateral tricuspid annular level of RV free wall in A4C assess the longitudinal dynamics of RV which was not well seen in visual scoring.

Same TDI measurements can also be obtained at left ventricular free wall at lateral mitral annulus level useful in assessing longitudinal dynamics of left ventricle in inferior wall myocardial infarction.

Significant RCA disease can be identified by assessing systolic tissue velocity of lateral tricuspid annulus in A4C view during dobutamine stress more preferably. MPI of lateral tricuspid annulus identifies proximal RCA lesions.

The Sm of tricuspid annulus decreased in patients with IWMI associated with RVMI than without RVMI with proximal RCA occlusion. (43-46).

Ozdemir et al in a study of 60 patients with inferior wall myocardial infarction RV MPI Value >0.70 showed proximal RCA occlusion with sensitivity of 78%and specificity of 91%.Our study also correlated with same sensitivity.

In the study of HSIAO SH et al RV MPI >0.42 has sensitivity 82% and specificity 76% in indentifying proximal RCA as culprit

vessel. Our study also correlated well with a sensitivity 72% and specificity 76%(p value <0.001).

MPI of lateral tricuspid annulus was more in RCA occlusion proximally than distal RCA occlusion.

In the study of HSIAO SH et al they mentioned Calculation of ratio of MPI of lateral mitral annulus with MPI of lateral tricuspid annulus reasonably predicts infarct related artery whether RCA if index is < 0.96 with sensitivity 80%,specificity73% and LCX if the index is > 1.06 with a sensitivity 71% and specificity 64%.Our study also correlated reasonably with this study in identifying proximal RCA if the ratio is <0.96 sensitivity 75% and specificity 77%,LCx if the value is >1.06 with sensitivity 75% and specificity 81%.

In the study of HSIAO SH et al systolic tissues velocity Sm of lateral tricuspid annulus was reduced in inferior wall MI associated with RVMI. Sm Value<11cm/s sensitivity80%,specificity81%. In another study by Ozdemir et al Tricuspid annulus Sm value <12cm/s sensitivity 63% and specificity 88% in identifying proximal RCA as culprit vessel. Our study also correlated reasonably with sensitivity 72% specificity 85% in identifying proximal RCA as culprit vessel

with tricuspid annulus Sm is <11cm/s. Beata Zaborska et al mentioned in their study of myocardial velocities in 101 patients if IWMI The SmRV < 12 cm/s as a cut-off for a diagnosis of RVMI had a 89% sensitivity and a 83% specificity

In the study of HSIAO SH et al they mentioned RV-IVRT >63 ms with a sensitivity of 73% and a specificity of 73% in identifying Proximal RCA occlusion. Our study shows RV IVRT >63ms in identifying proximal RCA culprit vessel with sensitivity 73% specificity 79%.

In a study by Yilmaz et al they indicated that tricuspid annular peak systolic and early diastolic velocities are reduced in patients with RVMI.(51)

The MPI otherwise known as Tei index commonly used in LV function assessment and it is non invasive Doppler measurement of ventricular function. Systolic time and diastolic time interval of MPI used in LV dysfunction determined and correlates with conventional parameters like LVEF and invasive measurement. (47)

These measurement has excellent reproducibility between observers and is useful simple method and is independent of heart rate and unaffected by geometry of ventricle. Tei et al also shows in patients with primary pulmonary hypertension right ventricular MPI is increased (0.93) and most important indicator to differentiate from healthy subjects.

Eldam et al also shown that in patients with Ebstein anomaly and severe right ventricular insufficiency RVMPI increases significantly.

We detected RVMPI > 0.41 in patients with proximal RCA occlusion and MPI index of lateral mitral annulur MPI / lateral tricuspid annulur MPI of > 1.06 in inferior wall myocardial infarction if lesion is isolated LCx and < 0.96 if occlusion is in proximal RCA with reasonable sensitivity and specificity.

Study Limitations :

Like most of the studies application of pulse tissue Doppler imaging in MPI assessment has its own limitation.

Although ventricular function depends on the contraction of myocardial fibres both in long and short axis. Long axis myocardial velocity only reflected in TDI. Contraction of circumferential fibres

does not affect myocardial velocities in the image in this localization. (22,23)

Global right ventricular function is not accurately reflected by TDI of lateral tricuspid annulus because of technical difficulties in imaging right ventricle.

Although infarct related artery is proximal to RV branch it remains imperfect determinant of RVI.

Lack of gold standard for diagnosing RV infarction is limitation of this study.

In tissue Doppler we can measure several velocities in several directions. Cardiac motion includs translational, rotational and deformational movement. Many tissues near the heart move due to transmitted motion of heart, pulsation of vessel, involuntary muscle movements and respiratory motion may interact with cardiac motion and leads to false Doppler shifts. With angle correction Doppler interrogations at one point determines the velocity of resultant of all these movements that is projected in the Doppler beam line.

There are movements in several axes in particular point and can never predict the resultant sum of vector.

The resultant vector accurately recorded if the Doppler beam is in the line of recording. (48) Abnormal findings are proportional to angle occurring between the transducer and the annular point of tricuspid motion.

One good point favouring tissue Doppler is the right ventricle function is dependant along the long axis. (49)

Parameters obtained by TDI provides quantitative data. Correction of these data with autopsy findings are the only gold standard is needed, that can be accepted.

CONCLUSION

1. Increase in right ventricular MPI more reliably predicts proximal RCA occlusion, though decrease of RV systolic tissue velocity and prolongation of time intervals like IVRT also predicts proximal right coronary occlusion in inferior wall MI.
2. Ratio of lateral mitral annulus MPI to lateral tricuspid annulus MPI reasonably predicts IRA in IWMI.
3. RVMI in association with IWMI can be diagnosed correctly with Tissue Doppler imaging since it is a prognostic indicator.
4. Tissue Doppler imaging could accurately predicts infarct related artery.

RECOMMENDATION

The results of this study indicates tissue Doppler imaging and MPI of tricuspid annulus and MPI index ratio between lateral mitral annulus and lateral tricuspid annulus reasonably predicts culprit vessel in inferior wall myocardial infarction.

Measuring RV Sm, RV MPI identifies proximal RCA occlusion which is a prognostic indicator of inferior wall myocardial infarction since it is associated with RVMI.

Assessment of systolic and diastolic function by MPI has greater diagnostic value.

In the era of coronary intervention knowing of infarct related artery by tissue Doppler imaging prior to coronary intervention is useful to select the hardwares needed for RCA or LCx artery PCI.

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ABBREVIATIONS

IWMI	-	Inferior wall myocardial infarction
RVMI	-	Right ventricular myocardial infarction
IVCT	-	Isovolumic contraction time
IVRT	-	Isovolumic relaxation time
ET	-	Ejection time
EF	-	Ejection fraction
RCA	-	Right coronary artery
LCX	-	Left circumflex
TDI	-	Tissue Doppler imaging
MPI	-	Myocardial performance index
IRA	-	Infarct related artery

PROFORMA

PROSPECTIVE STUDY OF Study on Tissue Doppler Imaging (TIM) in Acute Inferior wall Myocardial Infarction for identifying a culprit lesion.

Name :	Date of adm :
Age	Date of discharge/death
Gender	IP no.
Education	Ward/Unit
Marital status	
Occupation	Duration of hospital stay
Address	

Exclusion criteria

1. Doesn't opt for inclusion in the study
2. Death before angiogram and echocardiogram study
3. Absconded from follow up
4. Known prior M

- Date, time of symptoms
- Date, time of admission
- Time lag between symptoms & admission
- Drug name & form:

SYMPTOM PROFILE

1. Chest pain
2. Dyspnea
3. Palpitation
4. Giddiness
5. syncope

SIGNS

1. Tachycardia
2. Tachypnea
3. Hypotension /H
4. Oliguria
5. LV S3 &S4
6. Basal crackles
7. Pedal edema
8. Chest pain

9 Palpitation

10 Syncope

11. Dyspnea on exertion/ at rest

INVESTIGATIONS :

1. Blood sugar
2. Blood Urea
3. Serum creatinine
4. ECG (ST ST)
5. Echocardiogram

LVIDd	
LVIDs	
EF	

Tricuspid valve annulus		Mitral valve annulus	
S’		S’	
e’		e’	
a’		a’	
IVRT		IVRT	

IVCT		IVCT	
Ejection time		Ejection time	
MPI		MPI	
Mitral annulus MPI / Tricuspid annulus MPI			

Coronary Angiogram	LMCA	RCA	LCX	LAD

TREATMENT

1. Inj. Streptokinase
2. Inj. Heparin
3. Antiplatelets
4. T. Atorvastatin
5. ACE inhibitors
6. Beta blockers

OUTCOME

1. Identification culprit lesion is inferior wall myocardial infarction

ANALYSIS

1. Age
2. Gender
3. Drug given
4. Median delay between onset of chest pain and starting of treatment
5. Clinical / lab evidence of IW Mlin Echo

Ref. No. 23483 /E4/3/2013

Govt. Rajaji Hospital,
Madurai.20. Dated: . 02.2013

Institutional Review Board / Independent Ethics Committee.
Dr. N. Mohan, M.S., F.I.C.S., F.A.I.S.,
Dean, Madurai Medical College & 2521021
Govt Rajaji Hospital, Madurai 625020.
Convenor

Sub: Establishment-Govt. Rajaji Hospital, Madurai-20-
Ethics committee-Meeting Agenda- approval -regarding.

The Ethics Committee meeting of the Govt. Rajaji Hospital, Madurai was held at 11.00 Am to 1.00 Pm on 28.01.2013 at the Surgery Seminar Hall, Govt. Rajaji Hospital, Madurai. The following members of the committee have attended the meeting.

1.Dr. V. Nagarajan, M.D., D.M (Neuro) Ph: 0452-2629629 Cell.No 9843052029	Professor of Neurology (Retired) D.No.72, Vakkil New Street, Simmakkal, Madurai -1	Chairman
2. Dr.Mohan Prasad , M.S M.Ch Cell.No.9843050822 (Oncology)	Professor & H.O.D of Surgical Oncology(Retired) D.No.72, West Avani Moola Street, Madurai -1	Member Secretary
3. Dr.L. Santhana Lakshmi,MD Cell.No 9842593412	Associate Professor of Physiology/V.P Madurai Medical College	Member
4. Dr. Parameswari M.D (Pharmacology) Cell.No.9994026056	Director of Pharmacology Madurai Medical College	Member
5. Dr.Moses K.Daniel MD(Gen.Medicine) Cell.No 09842156066	Professor & H.O.D of Medicine Madurai Medical College	Member
6. Dr.D. Soundara Rajan,MS(Gen.Surgery) Cell.No 9842120127	Professor & H.O.D of Surgery Madurai Medical College	Member
7. Dr.Angayarkanni MD(O&G) Cell.No 9443567724	Professor & H.O.D of O&G Madurai Medical College	Member
8. Dr.P.V. Pugalenthi M.S, (Ortho) Cell.No 9443725840	Professor & H.O.D Ortho Madurai Medical College	Member
9. Dr. M. Sundarajan M.S., Mch Cell.No 9994924369 (Neuro Surgery)	Professor (Neuro Surgery) Madurai Medical College	Member
10 Thiru..Pala. .Ramasamy , BA.,B.L., Cell.No 9842165127	Advocate. D.No.72.Palam Station Road, Sellur, Madurai -2	Member
11. Thiru. P.K.M. Chelliah ,B.A Cell.No 9894349599	Businessman, 21 Jawahar Street, Gandhi Nagar, Madurai-20.	Member

The following Project was approved by the committee.

Dept of cardiology

Name of P.G.	Course	Name of the Project	Remarks
Dr.R.R.Saravanan	PG in DM (Cardiology) Madurai Medical College, Madurai-20.	Correlation of Annular MPI ratio of Tissue Doppler Imaging(TDI) in acute Inferior wall Myocardial Infarction with coronary Angiogram for identifying infarct Related artery culprit	Approved

Please note that the investigator should adhere the following: She/He should get a detailed informed consent from the patients/participants and maintain Confidentially.

1. She/He should carry out the work without detrimental to regular activities as well as without extra expenditure to the institution to Government.
2. She/He should inform the institution Ethical Committee in case of any change of study procedure site and investigation or guide.
3. She/He should not deviate for the area of the work for which applied for Ethical clearance.
She/He should inform the IEC immediately, in case of any adverse events pr Serious adverse reactions.
4. She/he should abide to the rules and regulations of the institution.
5. She/He should complete the work within the specific period and apply for if any Extension of time is required She should apply for permission again and do the work.
6. She/He should submit the summary of the work to the Ethical Committee on Completion of the work.
7. She/He should not claim any funds from the institution while doing the word or on completion.
8.She/He should understand that the members of IEC have the right to monitor the work with prior intimation.

Member Secretary **Chairman** **DEAN/Convenor,**
Govt. Rajaji Hospital,
Madurai-20.

To
The above PG students-thro' Head of the Departments concerned.

31/1/13

CORRELATION OF ANNULAR MPI RATIO OF TISSUE DOPPLER IMAING (TDI) IN ACUTE INFERIOR WALL MYOCARDIAL INFARCTION WITH CORONARY ANGIOGRAM FOR IDENTIFYING CULPRIT VESSEL

DISSERTATION SUBMITTED FOR

BRANCH – II
D.M. (CARDIOLOGY)

AUGUST 2013

THE TAMILNADU
DR.M.G.R. MEDICAL UNIVERSITY

CHENNAI, TAMILNADU

MASTER CHART

S.No.	Name	Sex	Age	SBP	RVMI	LVEF	**RV Sm**	RV Em	RV Am	**IVRT**	**IVCT**	RVET	**RV MPI**	LV Sm	LV Em	LV Am	IVRT	IVCT	LVET	**LV MPI**	**MPI Ratio**	IRA
1	Tanikodi	M	54	102	Y	45	9.6	7.6	11.4	78	96	234	0.74	7	7.8	6.3	40	38	218	0.35	0.48	PRCA
2	Deivamani	F	46	108	Y	40	7.8	7.4	9.1	92	70	285	0.56	6.7	6.9	5.9	53	47	285	0.35	0.62	PRCA
3	Balan	M	58	110	Y	48	8.29	6.3	12.5	96	46	246	0.57	6.6	8.29	10.6	43	57	254	0.39	0.68	PRCA
4	Adakkan	M	52	106	Y	42	8.8	11	11	88	85	275	0.62	6.1	11.7	6.04	55	42	232	0.39	0.62	PRCA
5	Pitchaiammal	F	54	112	Y	45	9.2	6.6	11.9	148	46	275	0.7	14	16	9.7	50	43	197	0.47	0.67	PRCA
6	Yeeraiah	M	50	122	Y	47	7.9	5.8	11	81	80	216	0.74	6.2	5.1	5.5	63	74	235	0.58	0.78	PRCA
7	Muthukrishnan	M	44	102	Y	46	7.8	4.4	10.1	80	84	216	0.75	7	9	8.7	64	52	276	0.42	0.56	PRCA
8	Koothoon Beevi	F	57	100	Y	45	10	11	8.8	74	69	239	0.65	6.3	15.7	6.8	67	53	310	0.38	0.65	PRCA
9	Balakrishnan	M	53	108	Y	38	10	7.5	13.5	67	88	243	0.63	6.7	4.4	8.2	43	57	236	0.42	0.42	PRCA
10	Panneerselvam	M	45	104	Y	40	9.2	8.3	15.9	94	85	214	0.83	7.8	8.1	8.5	41	53	241	0.39	0.46	PRCA
11	Irulayee	F	52	112	Y	43	8	9	14	201	67	236	1.13	7.1	10.5	8.1	74	99	298	0.58	0.51	PRCA
12	Muthusamy	M	58	120	Y	44	9.6	6.7	9.8	102	99	281	0.71	10.6	7.9	8.3	54	48	271	0.37	0.52	PRCA
13	Iqbal sherif	M	54	116	Y	45	8.9	7	10.2	77	70	204	0.72	10.9	9.5	7.5	42	50	215	0.43	0.59	PRCA
14	Sulochana	F	56	104	Y	47	9.5	11.2	14.6	120	85	234	0.87	7.6	6.6	9.6	43	47	230	0.39	0.44	PRCA
15	Sivasubramanian	M	58	102	N	52	12	11.6	10.9	55	58	289	0.38	9.3	13.5	9.1	70	74	275	0.41	1.1	DRCA
16	Selvaraj	M	54	100	N	36	12.8	10.8	8.5	61	129	278	0.6	9.2	10	10.4	92	85	264	0.67	1.11	DRCA
17	Kavitha	F	50	122	N	44	11.3	7.5	11	102	92	262	0.42	4.7	6.7	7.7	54	44	243	0.47	0.97	DRCA
18	Madasamy	M	53	114	N	36	12.6	6.7	8.6	58	35	261	0.35	4.29	3.8	6.4	67	80	220	0.66	1.88	LCx
19	Kamatchi	F	58	100	N	48	11.3	11.6	18.5	43	52	250	0.38	7.8	11.2	4.97	85	46	261	0.5	1.31	LCx
20	Dhanabal	M	56	106	N	42	11.6	8.4	13	42	70	264	0.4	6	9.1	6.5	67	85	282	0.51	1.26	LCx
21	Saraswathi	F	57	108	N	46	11.8	9.8	17.5	54	59	190	0.6	7.5	6.5	9.9	75	74	242	0.61	1.03	DRCA
22	Kulanthaivel	M	58	110	N	43	12	9.2	15.8	63	131	373	0.42	7.4	6.5	10.2	49	35	252	0.33	0.8	DRCA
23	Indira	F	51	116	N	45	11.5	9.4	16.6	128	92	407	0.54	7.6	5.9	10.2	87	90	262	0.67	1.25	DRCA
24	Ganesan	M	54	102	N	42	7.5	6.5	9.9	55	56	242	0.34	10.8	9.8	17.5	54	33	190	0.46	1.36	DRCA
25	Maairamam	M	55	100	N	45	7.4	6.5	10.2	54	51	252	0.42	10	9.2	15.8	100	94	373	0.52	1.23	DRCA
26	Vetrivel	M	58	108	N	42	7.6	5.9	10.2	62	115	262	0.67	10.5	9.4	16.6	128	92	407	0.54	0.8	DRCA

27	Kasthuri	F	56	112	N	44	12	11.6	10.9	55	58	289	0.38	7.5	6.5	9.9	55	56	242	0.44	1.15	DRCA
28	Muthu Irulandi	M	50	102	N	48	12.8	10.8	8.5	95	85	278	0.6	7.4	6.5	10.2	54	51	252	0.42	0.7	DRCA
29	Pitchaiammal	F	54	108	N	45	11.9	7.5	11	62	132	262	0.36	7.6	5.9	10.2	62	58	262	0.46	1.28	DRCA
30	Udhayakumar	M	58	110	N	45	11.6	9.4	16.6	128	92	407	0.34	10.5	9.4	16.6	58	60	380	0.31	0.98	DRCA
31	Abdul Jattar	M	44	102	N	42	7.5	6.5	9.9	55	56	242	0.37	7.5	6.5	9.9	55	36	248	0.37	1	DRCA
32	Sikkander	M	49	130	N	45	7.4	6.5	10.2	54	51	252	0.42	7.4	6.5	10.2	54	51	246	0.45	1.07	DRCA
33	Ramachandran	M	56	124	N	42	11.6	5.9	10.2	62	115	262	0.67	7.6	5.9	10.2	87	86	254	0.68	1.02	DRCA
34	Balasubramani	M	54	110	N	46	7.6	5.9	10.2	62	115	262	0.37	7.4	6.5	10.2	32	30	252	0.24	0.67	DRCA
35	Krishnamoorthy	M	52	126	N	44	12	11.6	10.9	55	58	289	0.38	7.6	5.9	10.2	87	60	262	0.56	1.76	DRCA
36	Poochendu	M	57	122	N	45	11.2	9.2	15.8	63	131	373	0.52	9.2	8.3	15.9	94	85	214	0.83	1.59	LCx
37	Chinnammal	F	55	118	N	46	11.8	9.4	16.6	128	110	360	0.38	8	9	14	44	41	236	0.36	1.2	LCx
38	Ramachandran	M	55	120	N	45	7.5	6.5	9.9	55	56	242	0.42	9.6	6.7	9.8	92	98	281	0.67	1.61	LCx
39	Madakulathoorn	M	57	110	N	45	7.4	6.5	10.2	124	100	280	0.38	8.9	7	10.2	38	31	204	0.34	0.9	LCx
40	Marudhan	M	58	110	N	43	11.6	5.9	10.2	137	104	262	0.34	9.5	11.2	14.6	40	30	234	0.3	0.89	LCx
41	Selvaraj	M	54	118	Y	43	9.2	8.3	15.9	94	85	214	0.83	10	9.2	15.8	100	94	373	0.52	0.62	PRCA
42	Parakkath Ali	M	54	120	Y	42	8	9	14	201	67	236	1.13	10.5	9.4	16.6	128	92	407	0.54	0.47	PRCA
43	Duraisamy	M	54	122	Y	42	9.6	6.7	9.8	102	99	281	0.71	7.5	6.5	9.9	55	56	242	0.44	0.61	PRCA
44	govindaraj	M	56	120	Y	44	8.9	7	10.2	77	70	204	0.72	7.4	6.5	10.2	54	51	252	0.42	0.58	PRCA
45	Veerakumar	M	56	114	Y	46	12.6	6.7	8.6	58	35	261	0.35	10.5	9.4	16.6	128	92	407	0.54	1.54	PRCA
46	Kannan	M	51	110	Y	48	11.3	11.6	18.5	43	52	250	0.38	7.5	6.5	9.9	55	56	242	0.44	1.15	PRCA
47	Raman	M	54	124	Y	45	6.3	15.7	6.8	67	53	310	0.38	8.3	7.5	11	102	92	262	0.52	0.99	PRCA
48	Pitchaiammal	F	53	122	Y	46	10.6	7.9	8.3	54	48	271	0.37	10.5	9.4	16.6	128	92	407	0.54	1.02	PRCA
49	Karuppasamy	M	54	112	Y	45	7.6	6.6	9.6	43	47	230	0.39	7.5	6.5	9.9	55	56	242	0.44	1.04	PRCA
50	Sekar	M	55	108	Y	45	12.4	6.7	8.8	58	33	261	0.35	9.3	13.5	9.1	70	88	275	0.57	1.03	PRCA